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A STUDY OF THE LESIONS IN A SECOND CASE OF TRAUMA OF THE CERVICAL REGION OF THE SPINAL CORD, SIMULATING SYRINGOMYELIA.*

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In this Journal in June, 1894, I reported two cases of traumatic affections of the cervical region of the spinal cord simulating syringomyelia. Both these cases occurred in men who had sustained severe injuries, involving fracture of the cervical spine, many years before. In both cases the resemblance to syringomyelia was marked. There were atrophy and paralysis of the shoulder and arm muscles, with fibrillary contractions, and spastic paraplegia without atrophy in the leg muscles, and without involvement of the bladder and rectum. Both patients had the dissociation symptom well marked. There was preservation of tactile sense, with loss of sensation for heat, cold, and pain; and, what was most striking, this dissociation symptom was unilateral. In one case, however, the paralysis was hemiplegic in type, and the dissociation symptom was entirely confined to the side opposite the paralysis. Thus the Brown-Séquard syndrome, modified, however, by the absence of tactile anesthesia, was presented; and this rare phenomenon of a crossed hemiplegia with the dissociation symptom, as a result of trauma, was one of the earliest instances that had been reported in the literature of the subject. The patient in this latter case having subsequently died, a full report of the autopsy was published in the spring number of *Brain*, 1898. In that paper it was shown that the lesion was confined largely to one side of the cord, and certain inferences were drawn from the findings as to the probable course of the various sensory fibers in the cord. The hemiplegia had existed on the side of the lesion; due, of course, to involvement of the lateral tract; while the involvement of sensation for heat, cold and pain on the side opposite the lesion was explained by the probable decussation of the fibers for these modes of sensation in the gray matter, and their subsequent course cephalad by way of Gowers' tract. The preservation of tactile sense was explained by the fact that the posterior columns were but little involved in the lesion. The case was a particularly favorable one for such a study, because the lesion was practically unilateral and involved almost the entire half of the cord, the part that had been the least injured being the posterior column.

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*Read at the twenty-fifth annual meeting of the American Neurological Association, June 14 and 15, 1899.

I have now the opportunity to report the results of the autopsy in the other of these two cases. Before doing so, however, I shall present a brief abstract of the clinical report from my preceding paper *(op. cit.)*.

James G., aged 55, a stone-cutter by trade, of good family and personal history, had met with two severe accidents. In 1876 he fell from a scaffolding and sustained, apparently, a fracture of the cervical spine. According to his own report his entire body was paralyzed, breathing was embarrassed, and urination, but not defecation, was affected. He stated that there was loss of sensation in the arms, but none below the waist; but probably not much reliance can be placed on this report of the sensory involvement. The essential facts are that he slowly recovered power—in the arms more slowly than in the legs—and was able at the end of two years to return to his work as a stone mason. His neck was deformed in a way that plainly indicated that the man had had a fracture of one or more vertebræ. The fact that he was able to return to work at all, and especially to such heavy work, is remarkable. That the cord had suffered injury in this first accident seems indubitable, and that this injury would have laid the foundations, in part at least, for subsequent degenerative changes, is probable, even if the man had not suffered a second accident. Five years after resuming work (*i. e.* in 1884) the patient had a second fall, this time from a ladder, a distance of fifteen feet, striking the back of his head and the spine between the shoulders. He was unconscious for six hours. This fall was followed by a return of the paralysis of the arms and legs, loss of control over the bladder, and obstinate constipation. According to the patient's own statement there was no marked loss of sensation, but we do not know how carefully that was determined. There was no priapism. There was severe pain at the seat of injury, and headache for three months. Power returned in the legs in two months, in the arms slightly by the end of seven months. After confinement to bed for four months the patient began to get about, but he was never able to work again after the second accident.

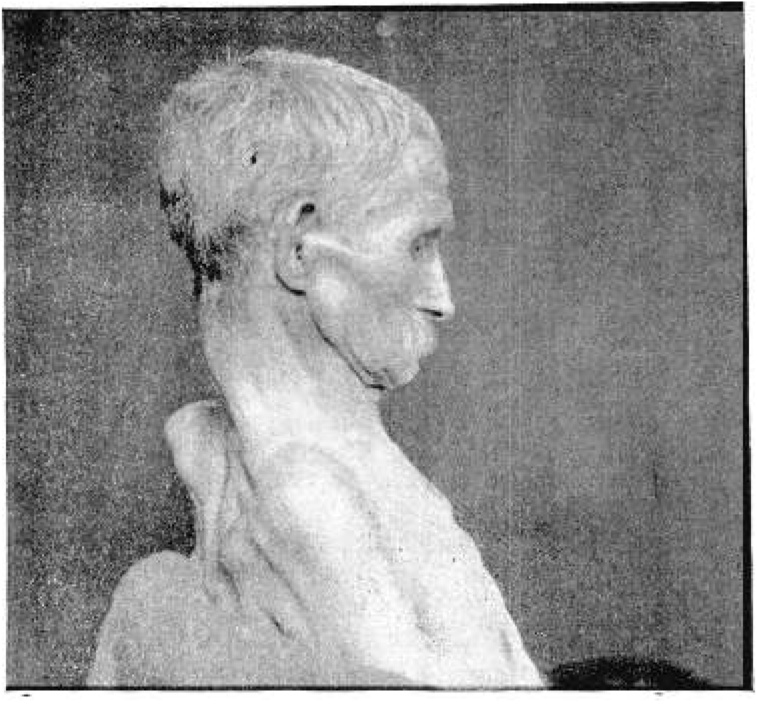

Fig. I.—Trauma of the cervical region of the spinal cord.

In 1894 the patient came under my care in the Philadelphia Hospital and continued there until his death in 1899. His condition when first seen was, in brief, as follows: There was advanced atrophy of the shoulder muscles of both sides, including the deltoid, pectoralis major, infra and supraspinati and the lower part of the trapezius; also the muscles of arms, forearms, and of almost all the muscles of the hands. Fibrillary contractions were active in some of the wasted muscles. The myotatic irritability was much increased. The arms were completely paralyzed, except for a slight power of flexion and extension of the forearm. The muscles responded to both currents; but there was decrease in excitability in the most wasted muscles, while there was hyperexcitability with duration tetany in others. There was no head-drop. The legs were paretic and contractured. The knee-jerks were exaggerated, and ankle-clonus was present. There was no muscular atrophy in the legs, and no trophic lesions, no bulbar symptoms, nor paralysis of the bladder. There was great deform-

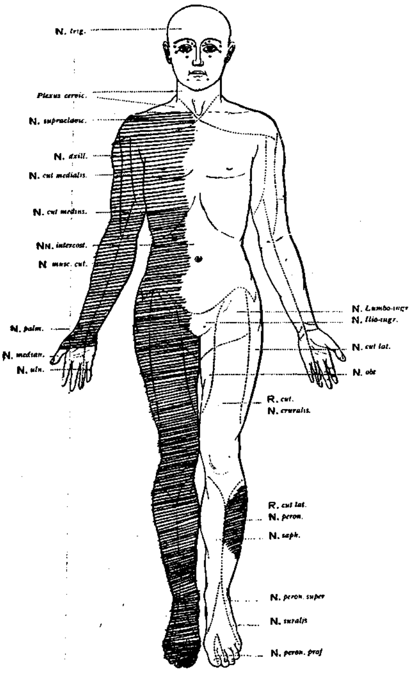

Fig. II.

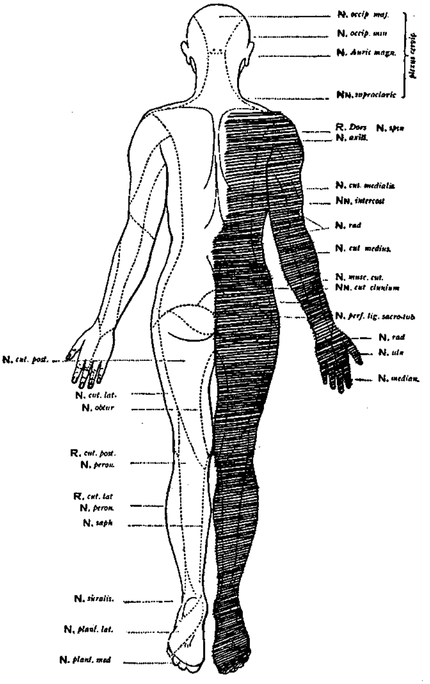

Fig. III.

Fig. II. Thermo-anesthesia and analgesia on right side. Tactile anesthesia in small area on left leg.

Fig. III. Thermo-anesthesia and analgesia on left side. Tactile anesthesia in small area on left leg.

ity of the cervical spine (see Fig. 1). The sensory symptoms were of great interest. There was still more or less neuralgic pain about the neck and occiput. Tactile sensation was good everywhere, as demonstrated by repeated tests. Thermo-anesthesia, or, more properly, anesthesia to cold, was marked on the right side as shown in the schemata. The patient distinguished heat better than cold, always calling cold hot. Analgesia was quite marked in some of this affected area, but was not coextensive with the anesthesia to cold. This condition remained unchanged for more than a year, during which time repeated observations were made.

The patient died in 1899, five years after the report of the case. During that time the symptoms remained practically unchanged. Death resulted from gradual exhaustion. Towards the end respiration became embarrassed, and this contributed directly to the death.

At the autopsy the cervical vertebræ, from about the 4th to the 7th were found much deformed and ankylosed, as the result of an old fracture. It was difficult to determine exactly what had been the extent of the injury, because of the state of ankylosis with formation of callus. As the chief interest of the case centers upon the injury to the spinal cord, I shall simply report the extent and appearances of the lesion in the spinal medullary tissue. There was some thickening of the dura mater overlying the seat of fracture, but it was hardly sufficient to be an important factor in the causation of the symptoms. The cord underlying the fracture was deformed and somewhat flattened. It was evidently extensively degenerated and sclerosed.

The microscopic appearances are as follows: At the point of greatest injury the cord is deformed and extensively degenerated. The gray matter is especially involved. It is difficult to distinguish clearly even the outlines of the anterior horns, and the multipolar cells seem to have almost disappeared. The posterior horns are more clearly discernible. The white matter is also widely degenerated. This is so especially of the antero-lateral columns; consequently the lateral pyramidal tracts, the direct cerebellar tracts and the antero-lateral ground bundles, with Gowers' tracts, all on both sides, are deeply involved. The only parts of the cord that seem to have maintained their integrity to any extent are the posterior columns, and to some extent the direct pyramidal tracts. This destruction of the gray matter and approximate preservation of the posterior columns are of great significance in view of the clinical phenomena presented by the case.

Above the seat of lesion the degeneration of the direct cerebellar tracts can be traced into the medulla. There is no degeneration of the fillet, nor of the pyramids.

Below the seat of lesion there is the characteristic descending degeneration of the lateral tracts. It presents nothing worthy of special note. The degeneration is complete as far as the tract can be followed into the lumbar enlargement, where it approaches the periphery as it diminishes in size. A point of special interest is the evidence of some slight and limited degeneration in the posterior columns below the cervical lesion. This is best marked in the cervical cord itself just below the seat of injury. The parts most sclerosed are the columns of Goll. The sclerosis exists as a wedge-shaped area, very similar to what is seen in ascending degeneration of Goll's columns in case of a transverse lesion in the lower dorsal or lumbar cord. As there is no lesion in this cord below the cervical region, this degeneration in Goll's columns cannot be properly an ascending one. It may possibly represent the degeneration of the descending sensory fibers after the dichotomous division of the entering neuraxon of the sensory neuron-body located in the posterior ganglion. Why it should so distinctly occupy Goll's columns to the exemption of the rest of the posterior columns is not apparent. The important fact remains, however, that even at the seat of greatest injury the posterior columns are the least injured of any portion of the cord, and this fact evidently has important relation with the clinical fact that the patient did not have impaired tactile sensibility. This slight degeneration in Goll's columns can be traced in diminishing intensity and extent as far as the dorsal region, where it is lost—a fact which argues against it being a true ascending degeneration. In the lumbar region the posterior columns—both Goll's and Burdach's—are absolutely intact. There is evidence in this cord of degeneration in the comma zones of Schultze.

In attempting to correlate the clinical phenomena in this case with the anatomical findings, I regard the following facts as conspicuous: There is extensive destruction of the gray matter and of the direct cerebellar tracts and Gowers' tracts, and comparative exemption of the posterior columns. This exemption of the posterior columns is, to be sure, not complete, but it is probably sufficient to explain satisfactorily the preservation of tactile sensibility. The inference, therefore, seems warranted, just as in the former case, that the tracts for heat, cold and pain pass up by way of the gray matter, eventually passing from the gray matter into Gowers' tracts; while tactile, or common, sensation is conveyed by the fibers in the posterior columns. Thus would be explained the analgesia and thermo-anesthesia—*i. e.* as due to the destruction of the gray matter; while the preservation of tactile sensibility is explained by the comparative integrity of the posterior columns. There is no cavity formation anywhere in this cord, but the seat of the lesion in the gray matter is the same practically as in most cases of syringomyelia.[1] In drawing this deduction I am in accord, in part at least, with some of the most competent observers. Van Gehuchten has summed up our knowledge on these points as follows: The fibers of the posterior columns, derived from the neurons in the posterior ganglia, serve for the conduction ot tactile sensibility; these fibers are direct, *i. e.*, not crossed, and terminate in the nuclei of the posterior columns; in other words, the nuclei graciles and nuclei cuneati. On the other hand, the impressions of heat, cold and pain are transmitted by way of the gray matter, through a second order of neurons, the cell bodies of which are located in the posterior horns and the neuraxons of which cross to the opposite side and pass cephalad by way of Gowers' tract. Van Gehuchten refers to the lesion of syringomyelia as demonstrating this probable course of the various sensory tracts. In my first case of trauma of the cervical cord (reported in *Brain*) this arrangement was clearly indicated by the fact that the lesion was almost entirely unilateral, and the thermoanesthesia was on the side *opposite* the lesion, while the tactile anesthesia and hemiplegia were on the *same* side as the lesion. In this respect that case was probably unique in the literature of this subject. In the present case the lesion is not unilateral, although the thermoanesthesia was confined to one side—a condition which may be explained by the fact that the clinical study was made some years before the patient's death, and, therefore, progressive degeneration may in the meantime have involved practically the gray matter on both sides.

In a volume of clinical lectures recently published, Brissaud[2] has illustrated this subject from some cases of spinal syphilis.

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[1] The microscopic sections were kindly made for me by Dr. David Riesman.

[2] "Lecons sur les mal. nerv." Deux. Série, Paris, 1899.

His explanation is practically the same as that given in the report of my case in *Brain*, in 1898. He especially emphasizes the possibility of the occurrence of the Brown-Séquard syndrome without loss of tactile sensibility—in other words, a hemiplegia with a crossed dissociation symptom. This, as already stated, was the exact condition in my first case. Brissaud, in order to illustrate his meaning, uses an ideal scheme, which almost exactly represents the real lesion in my case. Dejerine and Sottas[3] were among the first to record an instance of this crosed thermoanalgesia from a unilateral lesion, although the condition was not so striking as in my case, because there was thermoanalgesia of limited extent on the same side as the lesion also. The case was one of syringomyelia with a cavity almost limited to the right posterior horn; tactile sensibility was normal, but thermoanalgesia was present in the thorax and upper extremity on the side of the lesion and on the whole of the opposite side of the body. From the purely anatomical standpoint all such cases are in accord with the views of Edinger, who teaches that the sensory fibers which decussate in successive stages in the gray matter (the fibers here alluded to) do not decussate again in the medulla oblongata, but continue their course towards the cerebrum on the side opposite their entrance into the cord, and are rejoined by the fibers issuing from the nucleus gracilis and nucleus cuneatus—fibers which cross in the decussation of the lemniscus and constitute the arciform fibers.

In the latest (3d) edition of his book Van Gehuchten, summing up our knowledge of the course of the sensory fibers in the cord, concludes that the impressions of pain, heat and cold are transmitted by Gowers' tracts. As these tracts are made up largely of fibers arising from nerve cells in the gray matter on the opposite side, this conclusion is in accord with the pathological findings in my first case especially. In the present case the lesion in the gray matter is not sufficiently unilateral to permit this deduction, but the case at least proves that these modes of sensation are not conveyed by the posterior columns. Van Gehuchten inclines to adopt the results of Langendorff's experiments, which, he thinks, seem to prove that the posterior columns do not convey tactile impressions, but that these impressions must also pass through the gray matter, possibly reaching the brain by way of the direct cerebellar tracts. But such a conclusion, contrary to all clinical experience, does not seem to me warranted by Langendorff's experiments, which consisted in ligating the abdominal aorta (thus, it is claimed, invalidating the gray matter) and then noting alterations in the blood pressure upon irritating the skin—surely a method which takes much for granted. It seems, on the contrary, that we are still warranted in regarding the posterior columns as the paths for tactile, or common, sensation.

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[3] Bull. et Mém. de la Soc. de Biol., 25 Juil., 1891.

On the clinical side, which Van Gehuchten himself acknowledges is the only safe one for determining the sensory functions of the gray matter, several recent cases have been put on record. Among these is the case reported by Cushing,[4] in which a pistol shot wound of the spinal cord caused the dissociation symptom most marked on the side opposite the lesion and opposite the paralysis. The patient recovered, so, of course, the lesion could not be determined. Van Gehuchten[5] has recently contributed a paper on the dissociation symptom caused by traumata of the spinal cord.

10. SUR LES ALTERATIONS DES GRANDES CELLULES PYRAMIDALES CONSECUTIVES AUX LESIONS DE LA CAPSULE INTERNE (Changes in the Large Pyramidal Cells Resulting from a Lesion of the Internal Capsule). Marinesco (Rev. Neurol., 7, 1899, p. 359).

Six cases of hemiplegia were examined by Marinesco in which the internal capsule had suffered injury, with special reference to the large pyramidal cells of the cortex in the paracentral, frontal, and parietal convolutions. He found that the changes consisted in atrophy and degeneration of these cells analogous to the changes in the cells of the anterior horn after lesions and alterations in the peripheral nerves. The large pyramidal cells were the only ones affected, while the other cells of the cortex remained intact. Investigation of lesions more deeply seated and further removed, *e. g.*, in the pons or cord, showed that the further away from the cortex the pyramidal tract is injured, the later and less intense are the changes in the large cortical cells ("reaction at a distance"). JELLIFFE.

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[4] Am. Jour. Med. Sci., May, 1898.

[5] La Semaine Méd., 1899.